SLEEP QUALITY AND SYMPATHOVAGAL BALANCE DURING STRESS

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ABSTRACT

Sleep has been shown to be associated with the sympathetic and parasympathetic branches of the autonomic nervous system at rest and during stress. However, sleep has not been examined in the context of sympathovagal balance (the coupled relationship between the sympathetic and parasympathetic nervous systems) during stress. The current study investigated whether sleep quality was associated with sympathovagal activity and reactivity to stress. Female participants (N=59) underwent a psychosocial stress task and completed the Pittsburgh Sleep Quality Index while EKG and ICG data were collected. Sleep quality was associated with pre-ejection period at rest and during stress, ps<.05, but was not associated with high-frequency heart rate variability or with sympathovagal reactivity, ps>.05. These findings suggest that sympathetic activity and reactivity account for a significant amount of variance in the relationship between sleep and the autonomic nervous system.

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INTRODUCTION

The way a person responds to stress during the day is associated with how that person sleeps at night. To better understand the nature of this association, research has investigated links between sleep and a number of daytime psychological and physiological stress responses, including changes in nervous system functioning. For example, Mezick and colleagues (2014) identified a positive association between sleep duration and parasympathetic nervous system (PSNS) activity during a stress-inducing task. Another recent study found that sympathetic nervous system (SNS) activity during stress was negatively associated with sleep duration and sleep quality (Bagley & El-Sheikh, 2014). These and a handful of other studies have focused either on associations between sleep and SNS or sleep and PSNS (e.g., Martikainen et al., 2011; Palesh et al., 2008). Research has not yet examined associations between sleep and concomitant activity in both branches of the autonomic nervous system (ANS), often referred to as sympathovagal balance.

Stress responses and sleep are independently related to health (e.g., Ming et al., 2004; Mullington, Haack, Toth, Serrador, & Meier-Ewert, 2009; Pilcher, Ginter, & Sadowsky, 1997) and both are, at least in part, mediated by the ANS (e.g., Berntson, Cacioppo, & Fieldstone, 1996; Burgess, Trinder, Kim, & Luke, 1997). So, there are health implications to understanding the associations among sleep, sympathovagal balance, and stress responses. That is, an effect of sleep on sympathovagal balance may moderate the adverse effects of stress responses on health. Also, certain patterns of daytime sympathovagal balance may help identify individuals with unhealthy sleep patterns. This study is intended to evaluate the associations among sleep, stress, and daytime parasympathetic and sympathetic nervous system activity at rest and in response to stress (Franzen et al., 2011; Mezick et al., 2014).

Sleep and health

A proper amount of sleep is believed to have a restorative effect on the body, including the elimination of waste products from the brain (Xie et al., 2013) and improving physiological recovery from stress (Brindle & Conklin, 2012). Insufficient sleep, however, has been associated with adverse health including increased risk of diabetes, obesity, hypertension, cardiovascular events (Altman et al., 2012; Grandner, Chakravorty, Perlis, Oliver, & Gurubhagavatula, 2014), and mortality (Tamakoshi & Ohno, 2004; Wolk, Gami, Garcia-Touchard, & Somers, 2005). Although getting 7 to 10 hours of sleep is associated with lower risk of mortality compared to getting fewer than seven hours (Wolk et al., 2005), only about half of adults in the United States sleep seven hours or more (National Sleep Foundation, 2005). Moreover, approximately 42% of young adults complain of insomnia (Chen, Gelaye, & Williams, 2014), a disorder characterized by elongated sleep onset latency, disturbed sleep maintenance, and poor sleep quality, which have each been associated with increased mortality (e.g., Vgontzas et al., 2010).

Sympathovagal balance

Historically, the functioning of the ANS has been conceptualized as a reciprocal, coupled relationship between the SNS and PSNS branches. Indeed, their respective nicknames, the fight-or-flight system (SNS) and the rest-and-digest system (PSNS) suggest opposite ends of a continuum. So the conception has been that when the SNS is activated, the PSNS is simultaneously inhibited, and vice versa. However, recent research supports a model wherein the SNS and PSNS function independently of each other (Berntson et al., 1996); both can be simultaneously activated or inhibited, or one could be activated or inhibited while the other remains unchanged. This complementary relationship is called sympathovagal balance.

While there are a number of common measures of the combined effects of the SNS and PSNS (e.g., heart rate, respiration rate, blood pressure), non-invasive, independent measurements of the SNS and PSNS are less common. The SNS and PSNS simultaneously affect many major physiological systems and methods of independent measurement are not readily apparent. Nevertheless, researchers have identified and validated parameters that are independently affected by SNS and PSNS activity.

Heart rate variability (HRV) is the normal variation in time intervals between heartbeats. Variability in inter-beat-intervals (IBIs) within a high-frequency (HF) range of 0.15 to 0.40Hz is solely mediated by PSNS activity. HF-HRV is derived from EKG using fast Fourier spectral analyses. Increases in HF-HRV (i.e., greater HF-variability of IBIs) indicate increases in PSNS activity (e.g., Mezick et al., 2014).

Cardiac pre-ejection period (PEP) is mediated solely by the SNS. PEP is the time interval between the nervous system electrical signal stimulating the left ventricle of the heart and the initiation of the pumping action of the heart moving blood from the ventricle into the aorta. Using an electrocardiograph (EKG) signal and non-invasive impedance cardiography (ICG), which measures changes in thoracic density due to the pumping of blood into the aorta, we can accurately derive PEP. Decreases in PEP (i.e., shortening of the interim period) indicate increases in SNS activity (e.g., Berntson et al., 1996).

Sleep and sympathovagal balance

During sleep, there are consistent changes in sympathovagal balance. Notably, PSNS activity gradually increases from early transitional sleep stages (i.e., stage 1 and stage 2 sleep) to deep, slow-wave sleep (i.e., delta sleep) while SNS activity conversely decreases (Bonnet & Arand, 1997). Then, sympathovagal balance during sleep inverts when entering REM sleep

(Bonnet & Arand, 1997). Waking up from sleep is also marked by a general increase in SNS activity and corresponding decrease in PSNS activity (Burgess, Kleiman, & Trinder, 1999; Goff, Nicholas, Simonds, Trinder, & Morrell, 2010). Disruptions to sleep or insufficient sleep may interfere with ANS activity during sleep. This interference may then moderate daytime sympathovagal balance.

Stress, sympathovagal balance, and health

Like sleep, stress is associated with health outcomes. In general, chronic stress and repeated acute stress induce changes within the ANS that are associated with a range of negative health outcomes, such as thickening of arterial walls (i.e., atherosclerosis; Gianaros et al., 2005) and suppression of the immune system (Glaser & Kiecolt-Glaser, 2005; Padgett & Glaser, 2003). One hypothesized mechanism by which this happens is via gradual autonomic dysfunction (Sloan et al., 2005) or suboptimal sympathovagal balance (Sapolsky, 2004).

Stress has robust effects on the ANS. Specifically, stress activates the SNS or fight-or-flight branch of the nervous system. The SNS stimulates the release of the hormones epinephrine and norepinephrine, which stimulate β-adrenergic receptors, subsequently causing a host of fight-or-flight responses including increasing heart rate, elevating blood pressure, and facilitating the constriction or dilation of blood vessels in different parts of the circulatory system (Porges, 1992). These actions prepare the body to respond to a stressor. Concurrently, stress will inhibit the PSNS in order to reduce longterm processes, such as digestion, growth, or sexual reproduction. Post stress, PSNS activity will increase to facilitate recovery from stress by decreasing blood pressure, heart rate, and stress hormone release.

Sympathovagal responses to stress in different situations have been studied in laboratory settings by having participants perform a variety of stress-inducing tasks. Speech tasks, for

example, incorporate an element of social evaluative threat that, in general, activates the SNS and inhibits the PSNS (Gianaros et al., 2005; Mezick et al., 2014). Verbal mental arithmetic tasks lead to similar changes in sympathovagal balance (Berntson et al., 1996). On the other hand, tasks that demand cognitive attention instead of effortful responses, such as visual illusions, generally activate the PSNS yet do not affect the SNS (Berntson et al., 1996). Importantly, however, these patterns of sympathovagal activity are average responses and there is considerable variability among individuals. Also, after an acute stressor ends, both branches of the autonomic nervous system eventually return to their resting, baseline levels. The amount of time it takes for full recovery varies significantly among individuals (Linden, Earle, Gerin, & Christenfeld, 1997).

Research has found that changes in SNS and PSNS activity may link stress to health outcomes. It has been suggested that, independently, stress-influenced and sleep-influenced changes in the ANS may increase the risk of cardiovascular morbidity and mortality (Gianaros et al., 2005; Glos, Fietze, Blau, Baumann, & Penzel, 2014). In general, stress leads to an increase in SNS activity and a decrease in PSNS activity while sleep is associated with an increase in PSNS activity and a decrease in SNS activity. It seems likely that disturbance in either sleep- or stress-related sympathovagal balance will be associated with disruptions in the other.

Sleep, stress, and sympathovagal balance

A handful of recent studies has shown that sleep is associated with stress-induced changes in *either* the SNS or the PSNS. Mezick and colleagues (2014) assessed the association between sleep and PSNS activity during stress. They assessed sleep by means of actigraphy, a technique that quantifies daytime and nocturnal motor activity via accelerometers usually worn on the wrist (e.g., Martikainen et al., 2011), and used cognitive tasks and a speech task as

laboratory stressors. As reported in other studies of stress, PSNS activity decreased significantly in response to stress, as indexed by decreases in HF-HRV (Mezick et al., 2014). This was qualified by a significant interaction in which more total sleep time, measured by actigraphy, was associated with greater decreases in PSNS activity during the cognitive tasks but not during the speech task. Another study found that better sleep was associated with higher parasympathetic tone during mental math and speech tasks. In these studies sleep quality was indexed with several sleep parameters including sleep efficiency, number and length of wake episodes, and waking after sleep onset (Palesh et al., 2008).

There is minimal literature on the associations between sleep and SNS reactivity. One of the few studies found that increased SNS reactivity to stress, indexed by shorter PEP, was associated with fewer minutes spent sleeping, less sleep efficiency (a ratio of time spent sleeping to time spent in bed), and more fragmented sleep in preadolescents (Bagley & El-Sheikh, 2014). Another study, however, found no associations between sleep and PEP during stress (Martikainen et al., 2011).

Although these studies investigate associations between sleep and the ANS during responses to particular stressors, they are incomplete, opting to assess only one branch of the autonomic nervous system. Because the SNS and PSNS function independently and to varying degrees during specific stressors and among individuals, both branches should be independently accounted for. In this experiment we assessed both branches of the autonomic nervous system and to examine how sleep is associated with sympathovagal balance during stress.

In the proposed experiment, participants underwent a modified speech task based on the Trier Social Stress Test (TSST; Kirschbaum, Pirke, & Hellhammer, 1993), which has been shown to produce a robust psychophysiological stress response (Kudielka, Hellhammer, &

Kirschbaum, 2007). We recorded hemodynamic parameters before, during, and after the speech task using ICG and EKG. From these data we derived PEP and HF-HRV to respectively measure SNS and PSNS activity. By simultaneously measuring both branches of the ANS, we demonstrate how sympathovagal balance changes throughout the study. We also assessed self-reported sleep parameters, overall sleep quality, and health behaviors using the PSQI (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989) and a series of health questions, which participants completed following a recovery period.

This study aimed to clarify the associations between sleep quality and daytime sympathovagal balance at rest and in response to a psychosocial stressor. Although the literature pertaining to sleep and stress-induced SNS reactivity is scant and inconsistent, two studies gave us bases on which to make hypotheses (Bagley & El-Sheikh, 2014; Mezick et al., 2014). We hypothesized that poor sleep quality was associated with increased SNS reactivity to stress but not with PSNS reactivity. We also hypothesized that sleep was associated with daytime sympathovagal balance at rest (Bagley & El-Sheikh, 2014; Burgess et al., 1997) and during stress, but we did not have a specific hypotheses regarding the patterns of these associations (i.e., whether poor sleep quality would be associated with simultaneous SNS and PSNS activation, deactivation, or some other pattern).

METHODS

Overview

Data for this study were a subset of data from a larger study in which this candidate was the primary laboratory coordinator and experimenter. The candidate contributed to study development and generated the research questions addressed in the study presented here independent of the larger study.

Participants

One hundred twenty-one female undergraduate students from North Dakota State University participated in the parent study involving the effects of social support on Psychophysiology during stress. These students were recruited through an online signup system and received credit for their participation. Participants in the control (no support) condition (N=60) comprised the data for this study. One of these participants did not have sleep or physiology data and was therefore excluded from further analyses. The final sample included 59 female undergraduates. Participants were asked to refrain from extreme exercise, cigarette smoking, caffeine consumption, and eating a full meal for at least four hours prior to their experiment. Exclusion criteria included having a family history of cardiovascular disease, currently using medication, and being hypertensive.

Participant ages ranged from 18 to 30 years (M=19.14 years, SD=1.83 years). The majority of participants was in their first year of school (71.2%) and was full-time students (98.3%). The sample was predominantly White (78%), followed by Black (6.8%), Asian (6.8%), Hispanic/Latino (6.8%), and American Indian/Alaskan Native (1.7%).

Procedures

Participants arrived at the laboratory and completed an informed consent form. Then an experimenter directed the participant to take a seat and a research assistant attached electrodes measuring EKG and ICG with a six electrode configuration. Once clear signals were established, the experimenter left the room to collect a ten-minute resting baseline measure. Following the baseline measure, participants then completed a series of questionnaires associated with the parent study and answered questions about how they currently felt.

The participant was then given five minutes to prepare a speech on an assigned topic, which they delivered to two audience members. Participants were told that the audience members are experts in public speaking. A video camera was also set up to increase evaluation apprehension (i.e., stress). After five minutes of speech preparation, the experimenter and two female audience members entered the room. The audience members sat opposite the participant. The participant was instructed to begin her speech after a video camera was set to record by the experimenter.

During the speech, the audience members, who were confederates of the experiment, appeared to take notes. Like the experimenter, the audience members wore white lab coats and acted evaluative with occasional judgmental expressions. This protocol has been used in many past studies to reliably induce feelings of stress (Kirschbaum et al., 1993).

Following the speech task, participants completed a second questionnaire set, which asked about how they felt during the task. They then underwent an eight minute recovery period during which they were to “sit and relax”. After the recovery period, the electrodes were removed and a final questionnaire was completed. This questionnaire asked about health

behaviors, including sleep quality, and demographics. The experimenter then debriefed and thanked the participant.

Physiological measures

High-frequency heart rate variability (HF-HRV)

EKG data were collected using BioPac System (Goleta, CA) hardware, with two electrodes across the heart. Using MindWare Technologies software (Gahanna, OH), HRV was computed based on IBIs derived from the EKG. The software was used to identify artifacts (Berntson, Quigley, Jang, & Boysen, 1990) and to calculate HF-HRV using a fast Fourier transformation algorithm.

Pre-ejection period (PEP)

ICG data were collected with four electrodes on the front and the back of the neck, over the sternum, and on the ninth thoracic vertebrae (Cacioppo et al., 1994); thoracic density signals were derived from basal impedance data and change in impedance over time. MindWare Technologies Impedance Cardiography software (Gahanna, OH) calculated PEP based on the interim period between the Q-wave on the EKG signal and the B-point on the ICG signal (Cacioppo et al., 1994).

Psychological measures

Pittsburgh Sleep Quality Index (PSQI)

The PSQI (Buysse et al., 1989), a widely used measure in sleep research, asks questions pertaining to a number of subjective sleep parameters (see Appendix). Answers reflect usual sleep habits throughout the majority of the past month. Depending on the question, responses can be one of four items (i.e., not during the month, less than once a week, once or twice a week, and three or more times a week), a time (e.g., 10:30 PM), or a length of time (e.g., 7.5 hours).

Responses to the PSQI are computed into seven orthogonal domains. Each domain is assigned a value of 0 to indicate better sleep or a value of 3 to indicate poorer sleep. Sleep duration is computed by assigning values to ranges of hours of nighttime sleep (e.g., ≥7 hours receives a 0, <7 but ≥6 receives a 1). Scoring sleep disturbance entailed summing nine questions related to having trouble sleeping and assigning values based on the sum. To calculate sleep latency, we first assigned values to the ranges of how long it takes the individual to fall asleep and then assigned an overall value based on the first value plus the response to a question about getting to sleep within 30 minutes. We assigned values to a combined score of two questions querying trouble staying awake and maintaining enthusiasm to determine daytime dysfunction due to sleepiness. We calculated sleep efficiency by assigning values based on the quotient of nighttime hours of sleep divided by time in bed, using reported bedtime and waketime. The calculated domains of subjective sleep quality and needing medication to sleep were based on the scores of single items that respectively asked about overall sleep quality and the frequency of taking medication (prescribed or over-the-counter) to help procure sleep.

Finally, we summed the values of each domain to produce an overall score of sleep quality, which range from 0 (better sleep quality) to 21 (worse sleep quality). A score above 5 suggests the individual has poor sleep quality. This score also conceptually reflects the severity of the individual's sleep quality complaints. More information on the standard scoring of the PSQI can be found in the manuscript by Buysse and colleagues (1989).

There was one outlying response for sleep latency in minutes; however, when coded into the sleep latency domain score, this datum was no longer an outlier among responses. There was also one outlying response for bedtime; this response did not result in an outlying response when

calculated into the time in bed parameter. No participants were removed based on outlying PSQI responses, and there were no outlying global PSQI scores.

Health questionnaire

The health data were gathered using a health questionnaire that asked about various activities (e.g., caffeine use, exercise) performed the day of the study or over the past seven days. This questionnaire has been used in previous studies to screen for exclusion criteria (e.g., Hilmert, Christenfeld, & Kulik, 2002).

Positive and Negative Affect Schedule – Expanded Form (PANAS-X)

The PANAS-X (Watson & Clark, 1994) is a well validated self-report measure of present emotionality, featuring 60 words and phrases, each representing specific emotions or feelings (e.g., guilty, energetic). The participant rates how they are currently feeling each emotion on a scale from 1 (very slightly or not at all) to 5 (extremely). Subsets of these items combine to form more specific emotion composites (e.g., sadness, joviality) or general emotion composites (i.e., general negative affect and general positive affect; GNA and GPA, respectively). Participants completed the PANAS-X twice throughout the duration of the study: once before stress to assess baseline emotionality and again after stress to assess emotions during stress.

Task

Trier Social Stress Test (TSST)

Participants prepared a speech for five minutes and then delivered a five-minute speech in front of two expert female audience members. Speech topics varied and were assigned to participants. The experimenter remained in the room but off to the side.

Analysis strategy

Following collection of study data, the physiological data were scrutinized for artifacts using Mindware software and manual inspection by trained assistants. Then physiological data within different periods of the study were averaged. The baseline average consisted of the last five minutes of the ten-minute baseline period, allowing the participant to orient herself to the laboratory environment during the first five minutes of the period. The task average consisted of the five-minutes during which the participant delivered her speech. Consistent with past research, HF-HRV values were transformed using natural log to minimize skew (Task Force, 1996).

We used a variety of statistical analyses to examine whether sleep was associated with sympathovagal balance. First, we determined whether the speech task successfully induced stress in participants; we compared affect and autonomic physiology before and during stress using a series of paired samples t-tests. Second, we preliminarily evaluated associations among ANS activity and between sleep, ANS activity, and health-related variables by calculating descriptive correlations. Third, we visually conceptualized sympathovagal balance by plotting sympathovagal vectors. The sample was separated into groups of few or severe sleep quality complaints, and mean PEP and HF-HRV values for each group respectively comprised the x- and y-coordinates of the vectors. We plotted sympathovagal balance at rest, during stress, and the change between phases.

Fourth, we used regression analyses to examine the association between sleep and autonomic reactivity, as well as the association of sleep with the interaction between sympathetic and parasympathetic reactivity. We also used simple slopes analysis to further examine significant interactions between sleep and sympathovagal reactivity. Finally, we ran repeated measures ANOVAs to evaluate how sleep was related to the physiological changes throughout

the main phases of the study. Sympathetic and parasympathetic changes over time were separately examined between groups of few and severe sleep quality complaints.

RESULTS

Preliminary analyses: Stress induction

To test whether the stress task psychologically affected the participants as intended, we used a paired samples t-test to compare levels of general negative and positive affect before and during the stress task. Baseline levels of general negative affect (M=1.34, SD=.41) were significantly higher during stress (M=2.26, SD=.84), $t(58)$=-9.01, p<.001. Participant general positive affect (M=2.36, SD=.83) was significantly lower during stress (M=2.06, SD=.77), $t(57)$=3.49, p=.001. These differences suggest that the stress task successfully manipulated emotionality and induced psychological stress.

Physiologically, a paired test comparing baseline PEP (M=108.94 ms, SD=10.65) with task PEP (M=94.31 ms, SD=13.97), showed that PEP significantly decreased during stress, $t(44)$=8.56, p<.001, indicating an increase in SNS activity. Also, we found that levels of natural log transformed HF-HRV (baseline M=6.38, SD=.74) was lower during stress (M=6.24, SD=1.03), indicating a decrease in PSNS activity, although this difference was not statistically significant, $t(48)$=.91, p=.369. The combination of emotional and SNS changes brought on by the stress task are consistent with a typical fight-or-flight response to stress.

Correlations among autonomic nervous system activity

We calculated correlations among each branch of the ANS during baseline, task, and task minus baseline (reactivity) in order to identify physiological associations across the entire study period. Shorter baseline PEP (greater SNS activity) was correlated with shorter PEP during stress, r=.60, p<.001; shorter baseline PEP was also correlated with more baseline HF-HRV (greater PSNS activity), r=-.37, p=.013, change in HF-HRV from baseline to task, r=.39, p=.009, and marginally correlated with less HF-HRV during stress, r=.26, p=.082. Shorter PEP during

was correlated with more HF-HRV during baseline, r=-.42, p=.005, and less HF-HRV during stress, r=.32, p=.035, as well as with PEP reactivity, r=.67, p<.001, and HF-HRV reactivity, r=.54, p<.001. Greater baseline HF-HRV (greater PSNS activity) was marginally correlated with more task HF-HRV, r=.24, p=.095; HF-HRV reactivity was negatively correlated with HF-HRV during baseline, r=-.45, p=.001, and positively with HF-HRV during stress, r=.76, p<.001. PEP reactivity was marginally correlated with HF-HRV reactivity, r=.29, p=.060. The associations among physiological branches are generally consistent with a reciprocal relationship between SNS and PSNS activity before and during stress.

Correlations with health-related variables

Descriptive statistics and correlations among our primary variables of interest (PEP, HF-HRV, and PSQI) and health-related variables are presented in table 1. In previous studies our primary variables have been associated with various aspects of health. PSQI measures of sleep were not associated with any health-related activities performed *on the day of the study* (e.g., caffeine use, exercise), ps>.05, with the exception of individuals who reported taking prescription drugs the day of the study reporting spending more hours sleeping (M=7.53, SD=1.21) than those who did not (M=6.81, SD=1.23), F(1, 57)=4.56, p=.037, and individuals who ate breakfast the morning of the study reporting an earlier bedtime than those who did not, F(1, 56)=7.23, p=.009.

Activities in which participants engaged during the previous seven days, however, tended to be significantly correlated with various aspects of sleep. Notably, worse sleep duration scores were positively correlated with less alcohol use, p=.007; more hours spent sleeping was positively correlated with more alcoholic beverages, p=.022. Less sleep disturbance was correlated with more alcoholic beverages, p=.033. More time spent in bed was correlated with

more alcohol use, p=.003, which is congruent with time spent sleeping. Later bedtime was correlated with more servings of caffeinated beverages, p=.004, more smoking, p=.045, and less exercise, p=.029. Later waketime was correlated with more smoking, p=.031, and more alcohol use, p=.022. Additionally, longer PEP (less SNS activity) at rest was correlated with more exercise during the week before the study, r=.37, p=.010; other parameters of ANS activity were otherwise not correlated with any health-related variables.

Table 1. Correlations between PSQI domains and health-related variables

	Mean (SD)	hq3	hq5	hq7	hq11	hq12	hq16	hq18	hq20
Sleep duration§	.51 (.80)	.16	.06	-.35**	-.05	-.04	-.12	-.21	-.11
Sleep duration (hours)	7.05 (1.26)	-.17	-.04	.30*	.03	.04	.08	.20	.14
Sleep disturbance§	1.15 (.52)	-.02	-.08	.28*	-.18	-.13	-.37**	-.23†	-.22†
Sleep latency§	1.10 (.92)	.17	.11	.04	-.06	-.01	-.35**	-.05	-.27*
Sleep latency (min)	21.68 (19.91)	-.06	-.07	.01	-.11	-.18	-.11	.00	-.24
Daytime dysfunction§	.92 (.65)	.17	.04	-.03	-.26†	-.11	-.15	-.32*	-.15
Sleep efficiency§	.58 (.77)	-.03	.03	.07	.03	-.10	-.01	.11	-.01
Time in bed (hours)	8.24 (1.34)	-.16	-.02	.38**	.07	-.04	.01	.29*	.10
Bedtime	23:48 (77 min)	.37**	.26*	-.14	-.28*	-.15	-.37**	-.21	-.04
Waketime	08:02 (68 min)	.22†	.28*	.30*	-.23†	-.22†	-.40**	.11	.08
Needs medication to sleep§	.22 (.70)	-.17	-.09	-.10	-.08	-.02	-.14	-.16	-.31*
Overall sleep quality§	.97 (.64)	.29*	.21	-.05	-.33*	.03	-.35**	-.32*	-.31*
PSQI§	5.44 (2.87)	.15	.08	-.05	-.21	-.09	-.36**	-.27*	-.34**

Note. Sleep measures are calculated from the Pittsburgh Sleep Quality Index; § higher scores indicate worse sleep characteristics within that domain; hq3 = average servings of caffeinated beverages; hq5 = average number of cigarettes smoked; hq7 = average number of alcoholic drinks; hq11 = days of aerobic exercise; hq12 = days of anaerobic exercise; hq16 = days on which breakfast was eaten; hq18 = days on which fruits were eaten; hq20 = days on which vegetables were eaten; ***p<.001; ** p<.01; *p<.05; †<.1

Fewer breakfasts was correlated with higher sleep disturbance scores, p=.004, longer sleep latency scores, p=.006, later bedtimes, p=.004, later waketimes, p=.002, and higher PSQI global scores (indicating lower quality sleep), p=.005. Fewer days consuming fruit was correlated with more daytime dysfunction, p=.014, more time spent in bed, p=.026, and higher PSQI global scores, p=.041. Fewer days consuming vegetables was correlated with longer sleep latency scores, p=.040, needing medication to sleep, p=.016, and higher PSQI global scores, p=.008.

Individuals who restricted their food intake during the week before the study had longer sleep latency in minutes (M=41.75, SD=37.17) than those who did not (M=18.53, SD=13.85), F(1, 57)=11.04, p=.002. Individuals who binge ate during the week before the study had a higher sleep disturbance domain score (M=1.63, SD=.52) than those who did not (M=1.08, SD=.48), F(1, 57)=8.68, p=.005; a higher sleepiness-related daytime dysfunction domain score (M=1.50, SD=.54) than those who did not (M=.82, SD=.62), F(1, 57)=8.42, p=.005; and a greater change in PEP from baseline to task (M=-1.01, SD=1.32) than those who did not (M=.16, SD=.86), F(1, 43)=8.31, p=.006. Individuals who had a mood disorder (e.g., depression, anxiety) had worse sleep duration domain scores (M=1.20, SD=1.30) than those who did not (M=.44, SD=.72), F(1, 57)=4.36, p=.041; longer sleep latency (in minutes; M=45.00, SD=43.73) than those who did not (M=19.52, SD=15.28), F(1, 57)=8.46, p=.005; and worse sleep efficiency domain scores (M=1.40, SD=.89) than those who did not (M=.50, SD=.72), F(1, 57)=6.88, p=.011.

Correlations between sleep and autonomic nervous system activity

We calculated correlations between domains within the PSQI and measures of each branch of the ANS during the phases of the study in order to identify whether typical associations between sleep and ANS activity were present within our data (i.e., sleep is primarily

associated with SNS activity and not with PSNS activity; Burgess et al., 1997). Correlations among these study variables are presented in table 2.

Table 2. Correlations between PSQI domains and ANS parameters

	PEP			HF-HRV		
	Baseline	Task	Reactivity	Baseline	Task	Reactivity
Sleep duration (domain)§	-.08	-.24†	-.08	.02	.04	.04
Sleep duration (hours)	.08	.35*	.20	-.09	.02	.11
Sleep disturbance§	-.24†	.04	.16	-.15	-.14	.00
Sleep latency§	-.18	-.11	.15	.04	.02	-.05
Sleep latency (min)	-.22	-.14	.06	.04	.04	.02
Daytime dysfunction§	-.02	-.12	-.10	.07	.19	.08
Sleep efficiency§	-.16	-.01	.22	.04	.05	.05
Time in bed (hours)	.01	.35*	.30*	-.10	.05	.15
Bedtime	-.20	-.33*	-.09	.00	-.04	.00
Waketime	-.34	.06	.28†	-.11	.02	.19
Needs medication to sleep§	-.19	.15	.23	.10	.08	.01
Overall sleep quality§	-.18	-.37*	-.15	.05	-.04	-.15
PSQI§	-.25†	-.17	.11	.05	.06	.00

Note. Sleep measures are calculated from the Pittsburgh Sleep Quality Index; § higher scores indicate worse sleep characteristics within that domain; ***p<.001; ** p<.01; *p<.05; †<.1

Notable correlations include the association between hours spent sleeping and PEP during stress, p=.014, where more hours of sleep was associated with longer PEP or less SNS activity during stress, and the association between worse sleep quality and shorter PEP during stress, p=.010, suggesting that individuals who endorsed worse sleep quality had greater SNS activity during the task phase. However, sleep quality as measured by the final PSQI score was not associated with PEP during stress. Furthermore, worse PSQI global scores were only

marginally associated with greater baseline SNS activity (shorter PEP), p=.093. Domains within the PSQI were not associated with measures of HF-HRV, ps>.05.

Visualizing sympathovagal balance

To examine how sleep is associated with sympathovagal balance at rest and during an acute stress we initially inspected vector plots of sympathovagal balance at rest, during stress, and the change in sympathovagal balance from rest to stress (i.e., reactivity). We separated the sample into two groups: those who had few sleep quality complaints (PSQI scores ≤5; n=29) and those who had severe sleep quality complaints (PSQI scores >5; n=23). Ten participants (five from each group) were not included because they did not have both PEP and HF-HRV data due to equipment malfunction. In each figure, the x-coordinate represents mean PEP values and the y-coordinate represents mean HF-HRV values.

Figure 1 is a vector plot of mean sympathovagal balance of the two sleep groups at rest. We standardized (z-scored) baseline PEP and HF-HRV for the entire sample and then calculated average standardized values for each measure within each group. The plot in figure 2 depicts mean sympathovagal balance during stress, for which we also averaged the standardized task PEP and HF-HRV values for both groups.

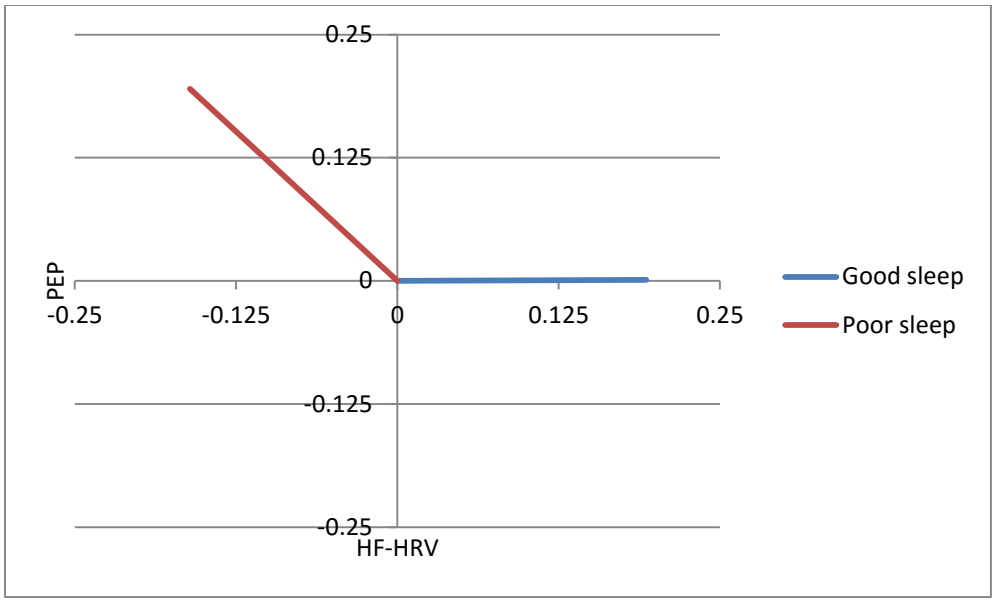

Figure 1. Sympathovagal balance at rest

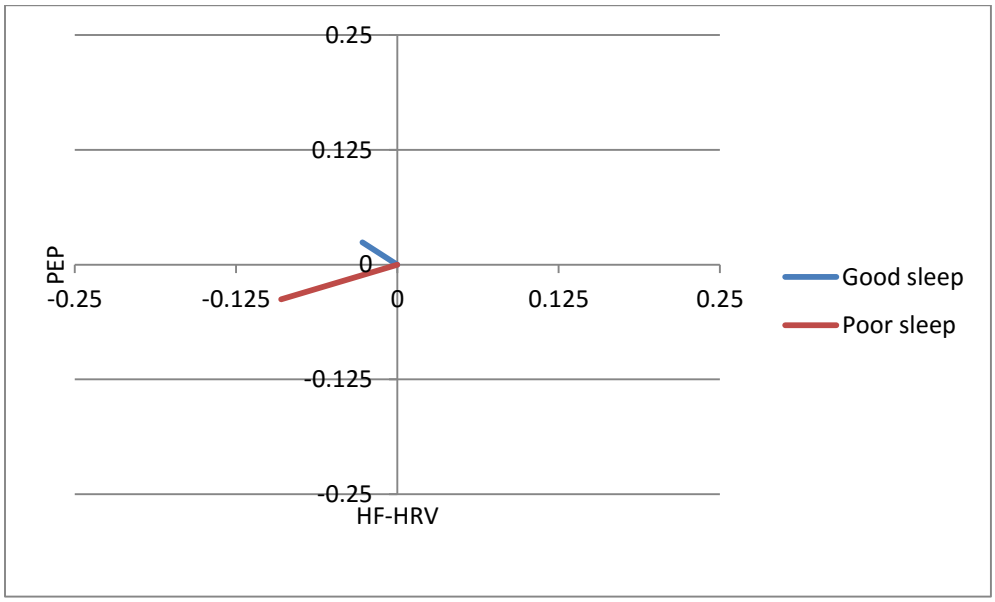

Figure 2. Sympathovagal balance during stress

The vectors of figure 3 denote standard deviations change in sympathovagal *reactivity* to stress. To calculate both PEP and HF-HRV reactivity, we subtracted baseline PEP and HF-HRV values from the respective task values. We then calculated the standard deviation of the reactivity scores for each index and divided each reactivity score by the standard deviation. We

averaged pairs PEP and HF-HRV reactivity values for each group. Figure 3 shows the zero-centered, standard deviational change of sympathovagal reactivity as a way to visually compare change in sympathovagal balance from baseline (0, 0) for sleepers with few sleep quality complaints and sleepers with severe sleep quality complaints.

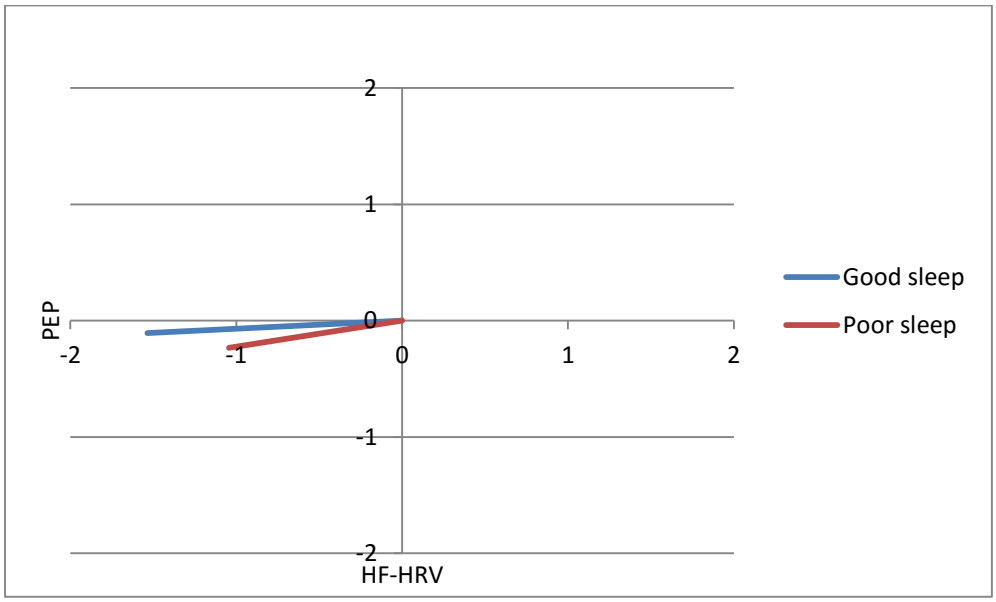

Figure 3. Recentered vectors

The vectors of few and severe sleep quality complaints show clear differences in sympathovagal balance at rest but not during stress. Casual inspection of figure 1 reveals that those who had severe complaints had increased ANS activity, demonstrated by concurrently decreased PEP and increased HF-HRV. This heightened profile suggests that individuals with severe sleep quality complaints experience a relatively high basal level of autonomic arousal.

Both groups of sleepers experienced a similar sympathovagal balance during the stress task, as shown by figure 2. Because of the differences in sympathovagal balance during rest, in order to achieve similar levels of sympathovagal balance during stress, the relative changes in the ANS of good and poor sleepers needed to differ. Inspection of figure 3 reveals this to be the case. Elevated basal sympathovagal balance in individuals with severe sleep quality complaints

limits how much the ANS can respond to stress, thus the shorter change vector compared to individuals with few sleep quality complaints. Moreover, the distinct baseline differences, taken with the insignificant differences during stress, suggest that stress may attenuate any differences between the two groups of sleepers during the actual stress response. There is a final sympathovagal balance that individuals reach during stress irrespective of where they begin.

Regression analyses

To test the primary hypothesis, we used regression analyses to examine the main effects and interaction effect of SNS and PSNS reactivities on sleep quality and sleep duration. Covariates that we controlled for (i.e., servings of caffeinated beverages, cigarette smoking, alcohol, exercise, food restriction, and binging eating) were ultimately chosen based on prior research that either found an association between the covariate and sleep or ANS activity (e.g., Léger, Poursain, Neubauer, & Uchiyama, 2008) or controlled for the variables themselves (e.g., Burgess et al., 1997; Grandner et al., 2014; Mezick et al., 2014).

Covariates were entered into step one. In step two we entered standardized baseline levels of PEP and standardized natural log transformed HF-HRV. To examine reactivity (changes in PEP and HF-HRV from baseline to task), we entered standardized levels of PEP and standardized natural log transformed HF-HRV during the speech task in step three. In the last step of the regression we entered a calculated interaction variable between standardized values of PEP reactivity and HF-HRV reactivity (baseline subtracted from task values). The interaction between PEP and HF-HRV reactivity was neither significantly associated with the PSQI global score, β=-.03, t(29)=-.13, p=.894, nor with self-reported hours of sleep, β=-.01, t(29)=-.03, p=.974. Moreover, the PSQI global score was significantly associated with baseline PEP, β=-.72,

t(29)=-3.13, *p*=.004, and PEP during stress, *β*=.51, *t*(29)=2.08, *p*=.047, but not with baseline HF-HRV, *β*=.04, *t*(29)=.23, *p*=.824, or HF-HRV during stress, *β*=.10, *t*(29)=.55, *p*=.587.

Table 3. Regression

	Pittsburgh Sleep Quality Index				
				Model 4	
	Model 1 *β*	Model 2 *β*	Model 3 *β*	*β*	95% CI
Constant	6.18***	5.62***	5.46***	5.46***	3.26, 7.66
Hq3	0.10	0.07	0.16	0.16	-0.20, 0.48
Hq5	0.04	0.06	-0.01	-0.01	-1.38, 1.29
Hq7	-0.20	-0.18	-0.31†	-0.31†	-1.72, 0.09
Hq11	-0.17	-0.17	-0.25	-0.25	-1.01, 0.28
Hq12	-0.12	0.02	0.13	0.13	-0.54, 0.94
Hq13	0.21	0.23	0.30†	0.30†	-0.10, 4.53
Hq14	0.16	0.22	0.34†	0.34†	-0.19, 5.56
Baseline PEP		-0.39*	-0.72**	-0.72**	-3.23, -0.67
Baseline HF-HRV		-0.04	0.04	0.04	-0.97, 1.20
Task PEP			0.51*	0.51*	0.02, 2.82
Task HF-HRV			0.10	0.10	-0.75, 1.31
PEP reactivity x HF-HRV reactivity				0.00	-1.07, 1.09
R^2	.16	.28	.41	.41	
F	.92	1.38	1.87†	1.66	
ΔR^2		.12	.13	.00	
ΔF		2.66†	3.23†	.02	

Note. hq3 = average servings of caffeinated beverages; hq5 = average number of cigarettes smoked; hq7 = average number of alcoholic drinks; hq11 = days of aerobic exercise; hq12 = days of anaerobic exercise; hq13 = whether food intake was restricted; hq14 = whether large quantities of food were consumed in a short period of time; *N*=42; ***p<.001; ** p<.01; *p<.05; †<.1

We also tested the association between a PEP and HF-HRV reactivity interaction and other sleep parameters using the domains that constitute the PSQI. There were two significant interaction effects:

How long it took participants to fall asleep (in minutes) was statistically significantly associated with the sympathovagal reactivity interaction, *β*=.36, *t*(29)=2.13, *p*=.042, so we

graphed the predicted values using the unstandardized coefficients. The interaction is depicted in figure 4. We evaluated the slopes using simple slopes analysis. The slope of PEP reactivity was significant at one standard deviation above the mean (high) HF-HRV reactivity, β=.67, t(31)=2.74, p=.010, but not at one standard deviation below the mean (low) HF-HRV reactivity, β=-.26, t(31)=-.85, p=.405. For those with high HF-HRV or PSNS reactivity to stress, higher PEP or SNS reactivity was associated with longer sleep latency than those with lower SNS reactivity. SNS reactivity was not associated with sleep latency when PSNS reactivity was low. Conversely, the slope of HF-HRV reactivity was not significantly associated with sleep latency at one standard deviation above the mean (high) PEP reactivity, β=.52, t(31)=1.59, p=.122, but was significantly associated with sleep latency at one standard deviation below the mean (low) PEP reactivity, β=-.48, t(31)=-2.13, p=.041. That is, when HF-HRV or PSNS reactivity was high, greater increases in SNS from baseline to task were associated with longer sleep latencies, but they were not associated when PSNS reactivity was low.

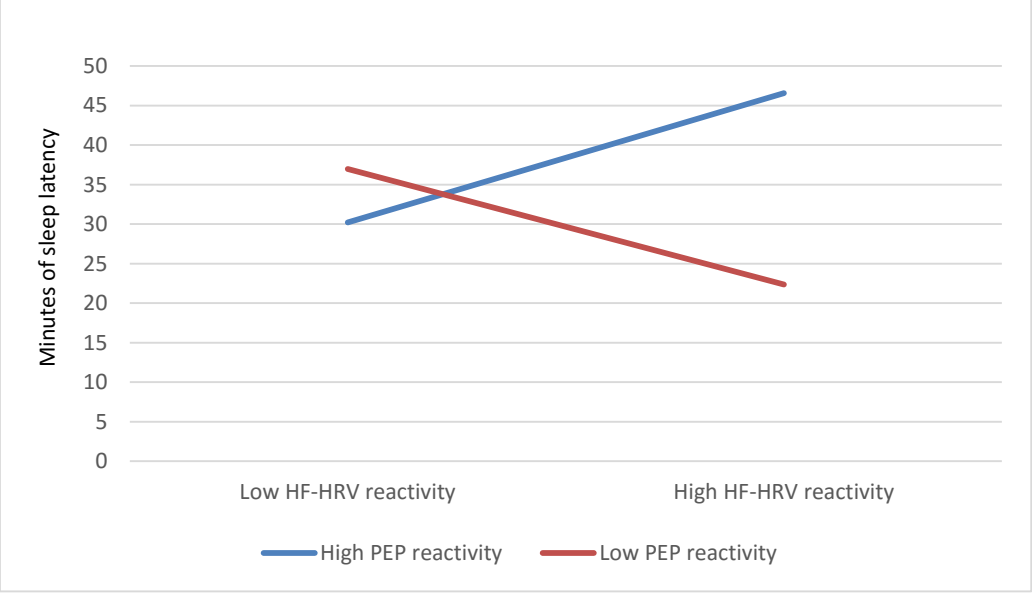

Figure 4. Simple slopes of sleep latency (in minutes)

The PSQI domain of daytime dysfunction due to sleepiness was also significantly, albeit marginally, associated with the interaction variable, β=-.36, t(29)=-1.90, p=.067. We graphed the predicted values for this interaction in figure 5. Simple slopes analyses revealed that the slope of PEP reactivity was not significantly associated at one standard deviation above the mean (high) HF-HRV reactivity, β=-.18, t(31)=-.67, p=.506, yet was significantly associated at one standard deviation below the mean (low) HF-HRV reactivity, β=.77, t(31)=2.23, p=.030. For those with low HF-HRV or PSNS reactivity to stress, higher PEP or SNS reactivity was associated with worse daytime dysfunction due to sleepiness than those with lower SNS reactivity. SNS reactivity was not associated with daytime dysfunction when PSNS reactivity was high. We found that the slope of HF-HRV reactivity was not significantly associated at one standard deviation above the mean (high) PEP reactivity, β=-.56, t(31)=-1.57, p=.127, and was marginally significantly associated at one standard deviation below the mean (low) PEP reactivity, β=.46, t(31)=1.90, p=.067. When PEP or SNS reactivity was high, PSNS reactivity was not associated with daytime dysfunction; however, when SNS reactivity was low, less PSNS reactivity was marginally associated with less severe daytime dysfunction.

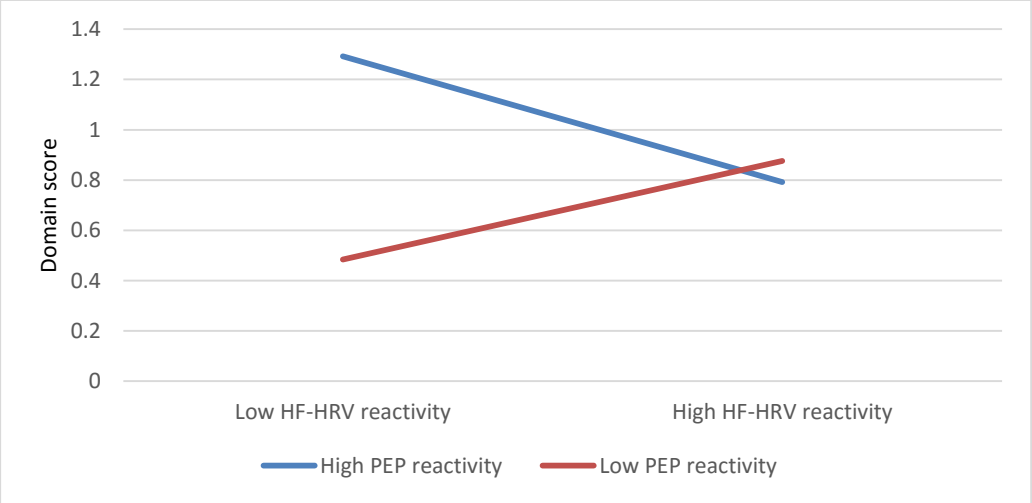

Figure 5. Simple slopes of daytime dysfunction due to sleepiness

Repeated measures ANOVA

We evaluated physiological changes throughout the study by running repeated measures ANOVAs separately looking at PEP and HF-HRV. The global PSQI score and hours of sleep were dichotomized for use as between-subjects factors within the same model. PSQI scores greater than 5 were considered severe sleep quality complaints, and scores less than or equal to 5 were considered few sleep quality complaints. Unstandardized measures of either PEP or natural log transformed HF-HRV for each main phase were entered as within-subjects variables.

The Mauchly's test of sphericity for PEP was significant, $X^2(2)=13.17$, $p=.001$, indicating that the assumption of sphericity had been violated. Degrees of freedom were corrected using Huynh-Feldt estimates of sphericity ($\varepsilon=.82$; Girden, 1992). There were main effects of phase on PEP, $F(2, 80)=48.63$, $p<.001$, $\eta p2=.55$, but no main effects of the PSQI global score, $F(1, 40)=1.08$, $p=.305$, $\eta p2=.03$. The interaction between PEP and the PSQI score was not significant, $F(2, 80)=.87$, $p=.405$, $\eta p2=.02$, suggesting that there were significant changes in sympathetic activity across the study phases, but these changes were not linked to sleep quality over the past month.

The Mauchly's test of sphericity for HF-HRV was also significant, $X^2(2)=3.77$, $p=.152$, indicating that the assumption of sphericity had been violated. We corrected for degrees of freedom using Greenhouse-Geisser estimates of sphericity ($\varepsilon=.72$). Main effects of phase on HF-HRV approached statistical significance, $F(2, 90)=3.05$, $p=.071$, $\eta p2=.06$, and there were nonsignificant main effects of the PSQI global score, $F(1, 45)=.21$, $p=.650$, $\eta p2=.01$. Additionally, there was a nonsignificant interaction between HF-HRV and the PSQI global score, $F(2, 90)=.21$, $p=.736$, $\eta p2=.01$. The results of this test suggest a pattern akin to

sympathetic activity across the study; that is, parasympathetic activity changed across the phases of the study yet was largely unrelated to sleep quality.

DISCUSSION

The goal of this study was to conceptualize the association between sleep and sympathovagal balance at rest and during stress. We set out to do this by first examining sympathovagal balance using a vector plot technique, which was used to illustrate differences in sympathovagal responses during different tasks by Berntson and colleagues (1996). For this study, we compared sympathovagal vectors of individuals with few sleep quality complaints to those with severe sleep quality complaints.

Inspection of the vector plots revealed that at rest those who had severe sleep quality complaints had a different pattern of sympathovagal balance compared to those who had few sleep quality complaints. Relative to their counterparts, sleepers with severe complaints had higher PSNS activity accompanied by higher SNS activity. The more activated SNS demonstrates a possible chronic fight-or-flight response in these sleepers. Consistent with this, we speculate that insufficient or poor quality sleep in this group elicited a minor, albeit persistent, stress response that elevated basal sympathovagal balance, despite being at rest.

Sleepers with few and severe sleep quality complaints demonstrated an identical pattern of sympathetic activation and parasympathetic withdrawal during stress. The absence of noticeable change in sympathovagal balance among sleepers who had severe complaints, however, was consistent with the suggestion that these individuals experience a chronic autonomic stress response. That is, those who had severe sleep quality complaints exhibited less of a change in sympathovagal balance to experimental stress, because they already had an autonomic fight-or-flight pattern of activity. This is further demonstrated by figure 3, which denotes greater change in SNS activity in sleepers with few complaints compared to sleepers with severe complaints. Although these vector depictions show patterns consistent with our

hypotheses, inferential analyses did not reveal statistically significant differences in the sympathovagal patterns of the two groups of sleeper.

We set out to statistically test if an interaction of sympathetic and parasympathetic reactivity to stress was associated with sleep quality complaints. We hypothesized that sleep quality would be associated with SNS reactivity but not with PSNS reactivity, and we tested this using a series of regression analyses. Our findings provided some support for this hypothesis. Sleep quality, as indexed by the PSQI, was associated with SNS activity but not with PSNS activity during stress. We also hypothesized that hours of sleep would be associated with sympathovagal balance at rest and during stress; however, PSQI-reported hours of sleep was neither associated with sympathovagal balance at rest nor during stress.

We further investigated the association between sleep and sympathovagal balance using repeated measures ANOVAs. Like the above vector plots, these tests compared sympathovagal activity of individuals who had few to those who had severe sleep quality complaints across the study. Sympathetic and parasympathetic activity both changed across the phases of the study, but neither interacted with sleep quality complaints over the prior month.

In spite of the interesting descriptive differences between sleepers in our conception of sympathovagal balance, the study did not statistically detect significant differences or an interaction between sleep quality complaints and ANS reactivity. Based on our regression analyses and repeated measures ANOVAs, sleep seems to be largely unrelated to stress-related sympathovagal balance; the two exceptions include sleep latency (in minutes) and daytime dysfunction due to sleepiness. We suggest some possible explanations below.

Our sample size was small. Having so few participants compromised the power of the study to detect small but potentially meaningful differences between sleep and sympathovagal

balance. There are also questions regarding participant stress responses in general. The study employed a speech task, which elicits psychophysiological stress responses (Kirschbaum et al., 1993; Kudielka et al., 2007) and, presumably, a normal autonomic stress response (e.g., Mezick et al., 2014). With respect to inducing psychophysiological stress, the study was successful. Participant affect changed significantly from baseline to task, suggesting psychological distress. Specifically, general positive affect decreased during the stress task, relative to baseline, while general negative affect increased. Along with these affective reactions, there were physiological changes in the ANS within the sample.

SNS activity increased from baseline to the stress task, which was expected and is typical for a normal stress reaction. PSNS activity concurrently decreased, but this decrease was not statistically significant. This nonsignificant change in PSNS activity contrasts with previous studies (e.g., Mezick et al., 2014), which exhibited significant decreases in PSNS activity. Although the trend of participant PSNS activity was consistent with prior research, its indistinct decrease in this study may suggest that participants did not exhibit a completely normal stress reaction.

The stress reaction exhibited by participants suggests that the results are contingent upon confounds introduced by the study design or inherent to the sample population. The speech task stress paradigm may not elicit the usual stress response or capture the variability of parasympathetic responses among all populations of college students (Hilmert & Kvasnicka, 2010). Alternatively, the exhibited sympathovagal balance may have been confounded by the amount of effort participants exerted during the stress task (Hilmert, Teoh, & Roy, 2013). For example, participants who exerted more effort may have consequently had a heightened sympathovagal balance relative to the profile of participants who did not exert much effort. The

latter may then have not activated their ANS as markedly as the former. Moreover, it may have been the case that individuals who had few sleep quality complaints were better able to engage with the stressor. That is, they were more alert and therefore more responsive to stress than participants who had severe sleep quality complaints.

Another plausible explanation for the nonsignificant findings is that sympathovagal balance is not associated with subjective sleep quality. We measured sleep using the PSQI, an index of self-reported sleep quality. Sleep quality itself is a subjective characteristic of sleep, which confounds its use as a measure of sleep. Unfortunately, this subjective component of sleep varies considerably from objective measures (e.g., polysomnography; Buysse et al., 2008; Grandner, Kripke, Yoon, & Youngstedt, 2006). The items that constitute the PSQI ask for estimates of sleep that could nonetheless be useful for investigation, from which an overall PSQI score is derived.

Contrary to its name, the score does not represent sleep quality, per se, because it does not purport to recognize the continuum from great to poor sleep quality. Scores of zero are possible, which creates a floor effect; the PSQI cannot characterize these lower scores as being anything other than not having complaints over sleep quality. Therefore, it becomes more reasonable to treat the PSQI as an instrument that measures sleep quality complaints, ranging from no complaints to many complaints, with a score of 5 still being a meaningful cutoff.

The PSQI asks about sleep during the prior 30 days. While this may detect individuals who have few and severe sleep quality complaints in general, it fails to detect acute sleep complaints, which may have a greater influence on sympathovagal balance and reactivity than more longterm sleep complaints. Perhaps future studies that employ the PSQI instrument could reduce the reference range to ask specifically about the prior week or even the prior night or two.

This would reveal whether an association exists between acute sleep complaints and sympathovagal balance.

Use of more objective measures would further increase the likelihood of detecting an association. Actigraphy would identify general sleep patterns and objective sleep quality for the duration leading up to the stress task. Polysomnography would reveal how sleep architecture is associated with sympathovagal balance. Objective measures of sleep would not quantify sleep complaints, still necessitating the use of a subjective measure, like the PSQI, but they would nonetheless increase and improve our understanding of sleep's relationship with sympathovagal balance.

Although this study had several limitations, it provides a meaningful addition to an otherwise limited literature on the topic of sleep and sympathovagal balance. Most previous studies examined the association of sleep with either SNS reactivity or PSNS reactivity but never both (e.g., Bagley & El-Sheikh, 2014; Mezick et al., 2014). Studies that investigated sleep and sympathovagal balance only examined the association with daytime sympathovagal balance at rest (e.g., Burgess et al., 1997), and those studies conceptualized sympathovagal balance orthogonally. That is, although they found a relationship between sleep and sympathovagal balance, the association was only with SNS activity; how sleep was associated with the interaction between SNS and PSNS activity was not examined. This is the first known study to investigate the interaction between both branches of sympathovagal balance with respect to sleep.

Further research into ANS activity and reactivity should incorporate the vector plot technique as a way to visualize sympathovagal balance. By comparing the sympathovagal vectors of sleepers at rest, during stress, and the change between the two, we identified that

differences between the two groups primarily exist at the basal level and not during acute stress reactivity. Sleepers who had few sleep quality complaints tended to have an activated ANS before even engaging with a stressor, but both groups exhibited a relatively normal autonomic response during stress. Although further analyses of these data revealed no significant differences between sleepers, the vector plots themselves revealed a potentially profound difference that could be detectable in a larger sample. Tangential and extension research ought to use vectors to examine potential differences until it is discernible that no differences exist.

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APPENDIX A. PITTSBURGH SLEEP QUALITY INDEX

INSTRUCTIONS:
The following questions relate to your usual sleep habits during the past month only. Your answers should indicate the most accurate reply for the majority of days and nights in the past month.
Please answer all questions.

1. During the past month, what time have you usually gone to bed at night?
BED TIME _____

2. During the past month, how long (in minutes) has it usually taken you to fall asleep each night?
NUMBER OF MINUTES _____

3. During the past month, what time have you usually gotten up in the morning?
GETTING UP TIME _____

4. During the past month, how many hours of actual sleep did you get at night? (This may be different than the number of hours you spent in bed.)
HOURS OF SLEEP PER NIGHT _____

For each of the remaining questions, check the one best response. Please answer all questions.

5. During the past month, how often have you had trouble sleeping because you . . .

a) Cannot get to sleep within 30 minutes

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

b) Wake up in the middle of the night or early morning

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

c) Have to get up to use the bathroom

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

d) Cannot breathe comfortably

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

e) Cough or snore loudly

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

f) Feel too cold

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

g) Feel too hot

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

h) Had bad dreams

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

i) Have pain

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

j) Other reason(s), please describe_____
_____

How often during the past month have you had trouble sleeping because of this?

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

6. During the past month, how would you rate your sleep quality overall?
   Very good _____
   Fairly good _____
   Fairly bad _____
   Very bad _____

7. During the past month, how often have you taken medicine to help you sleep (prescribed or "over the counter")?

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

8. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

9. During the past month, how much of a problem has it been for you to keep up enough enthusiasm to get things done?

No problem at all _____
Only a very slight problem _____
Somewhat of a problem _____
A very big problem _____

10. Do you have a bed partner or room mate?

No bed partner or room mate _____
Partner/room mate in other room _____
Partner in same room, but not same bed _____
Partner in same bed _____

If you have a roommate or bed partner, ask him/her how often in the past month you have had . . .

a) Loud snoring

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

b) Long pauses between breaths while asleep

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

c) Legs twitching or jerking while you sleep

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

d) Episodes of disorientation or confusion during sleep

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

e) Other restlessness while you sleep; please describe_____
_____

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

APPENDIX B. HEALTH QUESTIONNAIRE

Instructions: The present investigation provided measurements of heart rate and blood pressure, and therefore we want to identify factors which may affect these responses during the investigation. Please answer the following questions. All information that you provide will remain confidential, and feel free not to answer any questions that you feel uncomfortable in completing. If you have any questions as you go along, please ask the experimenter for clarification. Thank you.

Please answer the following questions regarding your behavior TODAY and THIS PAST WEEK, as indicated in the question:

1. **So far today**, how many cups of coffee (or 8-12 oz. serving of another caffeinated drink, i.e. cola) did you have? (indicate the number below)

_____cups of coffee or cola

2. In the **past HOUR**, have you had a cup of coffee (or 8-12 oz. serving of another caffeinated drink, i.e. cola)

1. YES
2. NO

3. Over the **past 7 days**, how many cups of coffee (or 8-12 oz. serving of another caffeinated drink, i.e. cola) have you had per day, on average?

_____ cups of coffee or cola

4. **So far today**, how many cigarettes have you smoked?

_____cigarettes

5. Over the **past 7 days**, how many cigarettes have you smoked per day, on average?

_____cigarettes

6. **So far today**, how many drinks containing alcohol (beer, wine, a mixed drink) have you consumed?

_____drinks containing alcohol

7. How often **over the past 7 days** have you had a drink containing alcohol (beer, wine, a mixed drink, any kind of alcoholic beverage)?

_____days

8. On days this **past week (7 days)** when you drank alcoholic beverages, how many drinks did you have all together on an average day? (By a drink, we mean a can or glass of beer, a 4-ounce glass of wine, a 1½ ounce shot of liquor, or a mixed drink with that amount of liquor).

_____drinks containing alcohol.

9. What was the most you had to drink in any **given 24-hour period** over the **past 7 days**?

_____drinks containing alcohol

10. **Today**, have you engaged in physical exercise, such as running, swimming, bicycling, tennis, fast walking, yoga, baseball, stretching?

1. No
2. Yes, for under 30 minutes
3. Yes, 30 minutes or more

11. Over the **past 7 days**, how many days did you engage in aerobic exercise: vigorous and continuous activity such as running, swimming, bicycling?

0	1	2	3	4	5	6	7

12. Over the **past 7 days**, how many days did you engage in anaerobic exercise: short burst of activity such as tennis, fast walking, yoga, baseball, stretching?

0	1	2	3	4	5	6	7

13. Did you greatly restrict your food intake over the **past 7 days**?

1. YES
2. NO

If yes, how many days this week did you restrict your food intake?

0	1	2	3	4	5	6	7

14. Did you binge at any time over the **past 7 days** (eat unusually large quantities of food in a very short period of time)?

1. YES
2. NO

If yes, how many days this week did you binge eat?

0	1	2	3	4	5	6	7

15. **Today**, have you taken any prescription drugs (including birth control)?

    1. YES
    2. NO

If yes, please list below:

16. **DURING THE PAST 7 DAYS**, how many days did you eat breakfast?

_____days this week

17. Did you eat breakfast **today**?

    1. YES
    2. NO

18. **DURING THE PAST 7 DAYS**, how many days have you eaten fruit?

_____days this week

19. Have you eaten fruit **today**?

    1. YES
    2. NO

20. **DURING THE PAST 7 DAYS**, how many days have you eaten vegetables?

_____days this week

21. Have you eaten vegetables **today**?

    1. YES
    2. NO

22. In the past **HOUR**, have you eaten any chips?

    1. YES
    2. NO

23. In the past **HOUR**, have you had any dairy products (milk, yogurt, cheese, etc.)?

1. YES
2. NO

24. Do you have any of the following medical conditions? Please read the list below and then answer yes (Y) if you have any of the conditions below. You do not need to indicate which of these conditions you have, just answer yes if anything on the list applies to you. If you do not have any of these conditions, please answer no (N).

_____ An endocrine disorder, such as Cushing's syndrome or Addison's disease

_____ An autoimmune disorder, such as lupus, rheumatoid arthritis, or multiple sclerosis

_____ A severe immune disease, such as HIV infection or AIDS

_____ A metabolic disease, such as adult diabetes, hypoglycemia, or hyperglycemia

_____ Chronic Fatigue Syndrome

_____ A diagnosed anxiety or depressive disorder (within last 6 months)

_____ A chronic infectious disease, such as hepatitis, tuberculosis, mononucleosis, etc.

_____ Any form of cancer or tumor

_____ A blood disease such as hemophilia or leukemia

_____ Serious allergies or asthma as an adult

_____ A cardiovascular condition, such as hypertension

_____ If you have been pregnant or breastfed in the last 6 months